

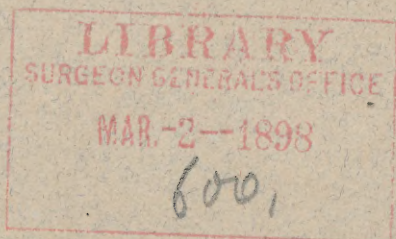
Porter (W. T.)

- I. On the Cause of the Heart Beat.
- II. The Recovery of the Heart from Fibrillary Contractions.
- III. Note on the Relation Between the Beat of the Ventricle and the Flow of Blood Through the Coronary Arteries.

BY

W. T. PORTER.

(Reprinted from the Journal of the Boston Society of Medical Sciences, No. 10, for March 30, 1897.)



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I. ON THE CAUSE OF THE HEART BEAT.

The methods employed by Wooldridge and Tigerstedt to isolate the ventricles from the auricles were incomplete, in that a part of the auricles containing nerve cells was left attached to the ventricles. The ligature used by Wooldridge and the atriotome employed by Tigerstedt cannot be applied in such a way as to entirely separate the auricles from the ventricles without compressing the coronary vessels in the auriculo-ventricular furrow, and thus interrupting the blood supply on which the continued action of the ventricles depends. The beat of the partially isolated ventricle of Wooldridge and Tigerstedt may therefore have been due to the discharge of motor impulses by the cells ~~in~~ the part of the auricles which, in their preparations, still remained attached to the ventricles.

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The following experiment illustrates a method which secures for the first time, so far as I am aware, the complete isolation of the mammalian ventricle.

Experiment of March 27, 1897.

A dog weighing 10 kilog, anæsthetized with morphia and ether, was bled from the left carotid artery, and the blood whipped, strained through glass wool, and diluted with an equal volume of 0.8 % normal saline solution. Normal saline of the same strength was meanwhile allowed to flow into the right jugular vein. After a short interval, the dog was again bled from the carotid artery. A second injection of saline solution was followed by a third bleeding. The product of these bleedings was mixed, and placed in a reservoir at the temperature of the body. The heart was now extirpated, a cannula tied into the ramus descendens of the left coronary artery, the interventricular septum and the auricles completely cut away, and all the ventricle removed except that portion supplied by the descendens itself. The cannula was then connected to a reservoir of warm blood-mixture, and the piece of ventricle perfused with the blood at a constant pressure, which, to begin with, was about 30 mm. Hg., but which was afterwards raised to 90 mm. Hg. The blood entering the cannula was bright arterial red. That emerging by the coronary vein was venous blue. In a few moments, the ventricle began to beat with

great vigor, shortening about seven millimeters in vertical diameter. An ordinary muscle lever magnifying 8 times, and weighted with 40 grammes, was fastened to a hook thrust through the apex, and recorded curves about 50 mm in height. The curve showed some irregularity both in force and frequency. The ventricle beat more rapidly when surrounded with blood at the temperature of the body than at room temperature, but the character of the contraction remained unchanged.

At 1 P. M., after writing curves for one hour, the ventricle was thrown into strong fibrillary contractions by stroking its surface with the electrodes of a Du Bois-Reymond induction coil, (tetanic stimulation). Five minutes later, good co-ordinated contractions returned. Forty minutes thereafter, the ventricle was thrown a second time into fibrillary contractions, from which it soon recovered.

Two and three-quarter hours after the ventricle began to beat, the experiment was broken off. The contractions were by this time very feeble, but still unmistakably co-ordinated.

Other experiments have shown that the right ventricle and the remaining portions of the left ventricle, namely, the septum and the circumflex area, will also beat if fed with blood through the coronary arteries.

It follows from these experiments that: (1) *The cause of the rhythmic contraction of the ventricle*

lies within the ventricle itself. (2) The cause of the rhythmic contraction is not a single localized co-ordination centre. The co-ordination mechanism, whatever it may be, is present in all parts of the ventricle. (3) The integrity of the whole ventricle is not essential to the co-ordinated contraction of a part of the ventricle.

It follows also that fibrillary contractions of the ventricle are not fatal. A summary of my experiments on recovery from fibrillary contractions will be given presently under a separate title.

I pass now to a refutation of the observation which is the chief reliance of those who believe that the heart beat is maintained by discharges from nerve cells, the observation, namely, that the apical half of the ventricle, even in the frog, does not beat spontaneously after its separation from the basal half.

Experiment of March 29, 1897.

The heart of a dog prepared as in the experiment of March 27th, was cut out, and a cannula tied into the ramus descendens of the left coronary artery not far from the apex of the left ventricle. That part of the apex which could be fed through the cannula was then excised. Both apex and basal portion fibrillated. The septum was removed. The piece of ventricle secured was 28 mm. in length (i. e. the direction

from the base to the apex), 23 mm. broad opposite the end of the cannula, and 27 mm. broad at the somewhat flattened tip of the apex. The ventricle measured from base to apex 70 mm. The part cut out was, therefore, the part which all observers, (except Berkley) believe to be lacking in nerve cells. The cannula was now connected with the blood reservoir and the apex perfused with blood. In a few moments regular and strong contractions set in. Curves were recorded with an ordinary muscle lever. During systole more blood escaped from the veins than during diastole. The experiment was stopped after the apex had contracted one hour and forty minutes. During a part of this time the preparation was in a bath of blood at the temperature of the body.

The experiment demonstrates that *the "apex" of the mammalian heart possesses rhythmic contractility*. Further, assuming that the general belief in the absence of nerve cells from the apical half of the ventricle is correct, the experiment demonstrates that *the rhythmic coordinated contraction of the ventricle is not dependent on nerve cells*.

II. THE RECOVERY OF THE HEART FROM FIBRILLARY CONTRACTIONS.

In experiments described in the previous communication ("On the Cause of the Heart Beat"), mention is made of the recovery of the dog's ventricle from

fibrillary contractions. The probability that the dog's ventricle would thus recover when supplied with blood was pointed out by me in earlier papers (*Centralblatt fur Physiologie and Journal of Experimental Medicine.*)

Fibrillary contractions have been seen in part of the ventricle, while other parts of the same ventricle were giving rhythmic, coordinated contractions.

The fact that the isolated apex and other isolated parts of the dog's ventricle will fibrillate, shows (1) that fibrillation is not caused by the stimulation of a localized centre for the coordination of the whole ventricle; and (2) that fibrillary contractions do not require nerve cells for their production, it being assumed that the general belief in the absence of nerve cells from the "apex" of the ventricle is correct.

III. NOTE ON THE RELATION BETWEEN THE BEAT OF THE VENTRICLE AND THE FLOW OF BLOOD THROUGH THE CORONARY ARTERIES.

At the meeting of the American Physiological Society in Boston, December 29, 1896, I pointed out that the isolated heart of the cat takes more blood through the coronary arteries from a reservoir under constant pressure when contracting than when at rest. The same fact was noted afterwards by Langendorff. When an isolated piece of dog's ventricle is fed through its coronary artery, the flow from the veins is seen to

be greater during systole than during diastole. On March 30, 1897, after having observed this phenomenon in several ventricles, I tied a cannula into the principal vein of a piece of the dog's ventricle consisting of the area supplied by the ramus descendens of the left coronary artery, the right ventricle and septum having been cut away except a narrow fringe near the descendens. The blood was clearly driven out of the cannula in the vein with each systole of the ventricle. The extent to which this pumping action of the ventricle influences the circulation through its walls and the form of the ventricular cavity during diastole ~~are~~^{is} now being studied.

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